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Scarlet fever and nineteenth-century mortality trends: a reply to Romola Davenport

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Romola Davenport's recent article is presented as a significant revision of the interpretation of the reasons for rising and then falling urban mortality in Britain in the nineteenth century put forward by Szreter and Mooney, which emphasized the importance of the politics of public health. Davenport's claims that mortality patterns *c*. 1830–70 were driven by a synchronized rise and fall of scarlet fever across Europe and North America, as well as in rural locations in Britain, are based on frail and inconclusive forms of evidence. The epidemiological evidence presented by Davenport in fact indicates a chronologically lagging—not leading—role for scarlet fever in contributing to the rise in urban death rates before 1850 and the subsequent fall in urban mortality after *c*. 1870 in Britain.

Romola Davenport's recent article argues that the chronology and geography of scarlet fever's incidence challenges the validity of a general interpretation of the causes of changing mortality patterns in the nineteenth century first put forward by Szreter in 1988, in opposition to the then-dominant 'McKeown thesis', and subsequently developed further by Szreter in 1997, by Szreter and Mooney in 1998, and by Szreter in 2005.[1]

Szreter and Mooney evaluated all the known, most robust demographic evidence available for Britain, *c*. 1780–1850, showing it testified consistently to a deterioration of mortality conditions in fast-growing industrial populations in the second quarter of the nineteenth century.[2] Modest improvement in the 1850s was not of great significance because there was no further improvement in the 1860s. It was the 1870s that truly heralded the commencement of a continuous mortality decline in both urban and national patterns. The general thesis developed to explain these trends drew on a reading of nineteenth-century public health and urban history to argue that political paralysis was the principal common cause of the deterioration, and that sustained improvement only became possible following long fought-for reforms to municipal electorates in the late 1860s.

Davenport now proposes an alternative thesis, which is that:

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[1] Davenport, 'Urbanization'; Szreter, 'Social intervention'; McKeown, *Modern rise*; Szreter, 'Economic growth'; Szreter and Mooney, 'Urbanization'; Szreter, *Health and wealth*.

[2] Szreter and Mooney, 'Urbanization'. A pattern re-confirmed by two subsequent studies of the industrializing towns of Sedgely and Wigan: Kitson, 'Industrialization'; Crompton, 'Industrialisation', fig. 4.1.

> The synchronized rise and fall of scarlet fever as a major cause of early childhood mortality between *c*. 1830 and *c*. 1870, in populations across Europe and North America, suggest very strongly that this was an autonomous biological phenomenon driven by changes in the virulence of the pathogen, albeit one that was propagated by networks of communication between international ports, and between ports and their hinterlands.[3]

Davenport sees this thesis and Szreter and Mooney's view as mutually exclusive, on the grounds that rural authorities in England and Wales and urban authorities in foreign states could not have experienced the same set of social and political developments with the same timing as industrializing towns and cities in Britain did.

Timing, however, is a fundamental obstacle to the acceptability and coherence of Davenport's counter-thesis. Charles Creighton, whose authority Davenport cites liberally, was quite clear that the 1840s marked the first of a series of serious and subsequently escalating scarlet fever epidemics in England, whose peak impact was in the 1860s.[4] Davenport's table 4 confirms that scarlet fever mortality throughout England and Wales in the three decades 1850–80 was much higher than even in three city populations in the 1830s and 1840s. Given that Woods has shown that scarlet fever mortality in cities was about four or five times higher per capita than in rural populations, table 4 implies that scarlet fever accounted for not more than 2 to 3 per cent of deaths among those aged 1–4 years in the 1830s and 1840s, but then rose to 13 to 15 per cent in the period 1850–80.[5] Both Woods and Wrigley et al. draw attention to a national deterioration in early childhood mortality appearing in the 1830s.[6] Scarlet fever, however, was not responsible for this.

Davenport's table 4 also confirms that there is no evidence that scarlet fever led the subsequent decline in early childhood mortality, which occurred from *c*. 1870 onwards. The extent to which scarlet fever caused deaths in early childhood in the 1870s was pretty much identical to that of the 1850s, whereas in the 1880s it fell precipitously to just 27 per cent of that level, and then to just 15 per cent in the 1890s.

The implausibility, on grounds of timing in relation to national trends, is compounded by the frailties in the more detailed evidence that Davenport presents. If scarlet fever were the principal cause of the mortality patterns in the eight non-urban locations assembled in Davenport's table 1, then we should see a rise in the early childhood mortality rate (ECMR) after the initial baseline row of figures for 1825–37, a sustained high ECMR in the subsequent decades, and then a decline in the rows of figures for 1881–90 and 1891–1900. However, only four of the eight locations show the substantial rise after 1825–37 (Dawlish fails to sustain its rise) and only one of the eight (Bottesford) shows a marked fall in 1881–90. ECMR falls in the previous decade in the other seven locations.

Indeed, the relative unimportance of scarlet fever in rural England is what one would expect from the geographical epidemiological analysis conducted by Woods. Woods showed that in the 1860s, the first decade for which we can have confidence

[3] Davenport, 'Urbanization', p. 480.
[4] Creighton, *History*, vol. 2, p. 727.
[5] Woods, *Demography*, pp. 324, 326.
[6] Woods, 'On the historical relationship'; Wrigley, Davies, Oeppen, and Schofield, *English population history*, pp. 259–61.

that the registration system was recording scarlet fever deaths reasonably accurately, the rural per capita death rates were four to five times lower than in urban districts.[7] Thus, the claim by Davenport that 'scarlet fever was an important cause of death in rural as well as urban populations' is not justified by Woods's data or analysis.[8]

Davenport's table 2 shows urban early childhood mortality patterns in 15 of the largest provincial cities. The highest rates mainly prevailed in 1830–50, alleviated somewhat in 1850–70, followed by a definite plunge to a lower level for 12 of the 15 cities from 1871 that was sustained to 1900. However, the trajectory of scarlet fever's incidence, as we have noted, was a sharp upward shift in 1850–80 until a dramatic drop in the early 1880s.[9] So, again, attention to timing indicates that scarlet fever did not initiate or drive these trends in urban early childhood mortality patterns.

Davenport's table 3 shows that urban mortality conditions in 1838–70 remained relatively unchanged, with an average life expectancy of 33 years, before improving in the 1870s. This contrasts with Szreter and Mooney, who found a rather lower life expectancy value of 29.5 years, as a weighted average of all the largest English provincial cities for the 1830s and 1840s, which then improved to a value of 34 years in 1850–70. They pointed out that that value was still below where such cities had been in the 1820s, if the unusually high quality evidence for the city of Glasgow was used as a guide.[10] It was argued that this was reasonable, because it was a city very much like a composite of Liverpool and Manchester, comparable in size, industrial complexion, and recent growth rates. It also registered a closely similar life expectancy trajectory to Liverpool and Manchester, in both 1830–50 and 1850–70.[11] Davenport presents newly calculated life expectancy values for 1838–44, which show Liverpool and Manchester Registration Districts (RDs) with higher values of 27 and 28 years than those of 26 and 25, respectively, in Szreter and Mooney's table 3. However, our table 3 figures (reporting Farr's contemporary figures) were not, as Davenport claims, our final estimates. They were then adjusted before further use to reflect the full 'administrative city' populations (beyond the RD), to values of 28 and 27, respectively.[12]

[7] Woods, *Demography*, pp. 323–4, 326.

[8] Davenport, 'Urbanization', p. 477.

[9] Despite the evidence of her tab. 4 and fig. 8, Davenport also states that scarlet fever's 'decline from the 1870s was a major driver of the early mortality decline among young children, and therefore made a very substantial contribution to the secular rise in life expectancy from the 1870s onwards'; ibid., p. 478. The principal citation here is to Hinde and Harris, 'Mortality decline', whose tab. 3, p. 383, does appear to show scarlet fever beginning to make a substantial contribution to improving life expectancy in the 1870s (though much less than that contributed by decline in the classic sanitation and hygiene diseases and somewhat less than respiratory tuberculosis). However, as Davenport's tab. 4 shows, the relatively high proportion of deaths at ages 1–4 attributable to scarlet fever in the 1870s is virtually identical to the proportion in the 1850s. It is because the rate was somewhat higher still in the 1860s that the methodology used by Hinde and Harris has registered this recession back to the still-high rates of the 1850s as a fall, which is contributory to the wider decline in mortality in the 1870s, driven mainly by a reduction in sanitation diseases. In fact there was no definite reduction in scarlet fever mortality from the heightened level prevailing across the whole period 1850–80 until a very rapid fall to a much lower level took place during the years 1881–5. With thanks to Bernard Harris for discussion on this point (personal communication, 5 June 2020).

[10] Szreter and Mooney, 'Urbanization', pp. 103–4.

[11] Ibid., pp. 96–9, 103.

[12] Ibid., p. 94. Davenport, 'Urbanization', p. 471, is therefore incorrect that values of 26 and 25 'informed [our] very low estimates of life expectancy in large towns'.

The selection of seven RDs representing the eight largest provincial cities (excluding Leeds) listed in Davenport's table 3 gives a mean of 33 years life expectancy for 1838–44. However, there are two problems with table 3. First, it includes Bradford, despite the fact that Szreter and Mooney very clearly warned that its high life expectancy values were a statistical artefact due to the RD's inclusion of a large surrounding area of Pennine villages and countryside.[13] The distorting effect of including Bradford is compounded by Davenport's choice of an unweighted mean, which is unusual in a demographic study since the cities of Manchester and Liverpool were each over twice the size of the other cities listed (apart from Birmingham which they also exceeded by about 30 per cent). A weighted mean would give a value of 32 in Davenport's table 3, and without Bradford a value of 31, which is much closer to Szreter and Mooney's mean value of 30 (29.5), which was calculated for 'administrative cities' (not just RDs), and so is likely to provide both more accurate figures and a more reliable basis of comparison with the Glasgow figures for 1820–40.

The Glasgow administrative city data are of more value to demographic historians of urban Britain in this period, 1820–40, than anything else available before the improved operations of the 1841 and 1851 censuses and the civil registration system under William Farr. This is because we know in detail how the Glasgow censuses of 1821 and 1831 were conducted and how the data on baptisms and burials were collected, verified, and checked under the direction of James Cleland.[14] Cleland knew, for instance, that the public registers of baptisms were highly defective and he also knew exactly by how much, because of the extremely careful supplementary procedures he put in place, which he also diligently documented.[15] It is due to his thorough methods—and our knowledge of them—that we can have confidence in the data he produced, which consequently attracted the contemporary attention of T. R. Edmonds, the leading exponent of life table techniques—from whom Farr learned his trade—who averred of the Glasgow data that, 'These materials are far superior to any previously existing in Britain; the information is supplied in a perfect form, and the accuracy of detail is unimpeachable'.[16] This means that the age-specific death rates Edmonds compiled from Cleland's data for Glasgow in the 1820s and 1830s, which were the basis for Szreter and Mooney's estimation of the detailed trend in life expectancy from 1821 through to 1841, are clearly the best urban evidence we have.[17] For the same reason, we also placed very considerable value on the Carlisle data collected for 1779–1813 by John Heysham, whose investigative techniques Cleland learned from; and whose data was used by the pioneering actuary Joshua Milne for the famous Carlisle life table that he published in 1815.[18]

Regarding the international evidence, Davenport's argument is that relevant measures of mortality can be found showing a pattern of synchronized rise and fall in cities all across Europe including Russia, and North America, which supposedly indicate the importance of a trans-Atlantic scarlet fever pandemic. By contrast with

[13] Szreter and Mooney, 'Urbanization', pp. 90–1, n. 18.
[14] Flinn et al., *Scottish population*, pp. 72–6, 83–4, 376–8.
[15] This was described in some detail in Szreter and Mooney, 'Urbanization', pp. 95–7.
[16] Edmonds, 'On the mortality at Glasgow', p. 353.
[17] Szreter and Mooney, 'Urbanization', tab. 5.
[18] Milne, *Treatise*.

the Glasgow and Carlisle evidence, many of these data are of highly questionable validity. Very few relate directly to records of scarlet fever, and in fact not many relate to early childhood mortality.

The evidence for Prussia and Russia can be summarily discounted as it refers to crude death rates, which are notoriously unreliable for evaluating the timing of mortality trends in growing cities. Where the cited evidence does relate to measures derived from age-specific data, it offers virtually no support for an international deterioration in urban mortality in 1830–50, driven by a pandemic of a virulent strain of scarlet fever. None of the three French cities studied by Preston and van de Walle support this interpretation. Davenport acknowledges that Lyon does not fit the pattern; but it is not acknowledged that neither does Marseilles nor Paris.[19]

Stockholm, another city with figures cited by Davenport in fig. 7b, exhibits a definite, marked rise in ECMR; however, this is not in the period 1830–50 as in Britain, but instead in the period 1850–70. Davenport accepts that the age-specific Canadian data that are available do not support a claim for a mortality deterioration among the young in the 1830s and 1840s.[20]

Evidence is cited for Belgium, indicating a mortality crisis not in 1830–50 but later, in 1850–70. Dutch and north Italian evidence is mentioned but this, too, fails to conform to the British pattern of marked urban deterioration in 1830–50 and improvement from the 1870s. In the well-documented case of Amsterdam, life expectancy at birth hovered around 30 years *c*. 1800–60 and then suddenly improved in the 1860s to values above 35 years.[21]

So, there is in fact very little demographically robust evidence adduced by Davenport to support the claim that there was a mortality crisis specifically in the 1830s and 1840s across Europe, Russia, and North America, that can be claimed as evidence that Britain's urban mortality crisis in the second quarter of the nineteenth century was part of a trans-Atlantic pandemic impact of a newly virulent variant of *Streptococcus pyogenes*.

Furthermore, there is the additional problem that in fact the major impact of scarlet fever in Britain did not occur in the period 1830–50 when the urban mortality crisis struck. Positive evidence presented for significant scarlet fever mortality before the 1840s is slight. In Davenport's table 4 there are data for Manchester, Glasgow, and Perth showing a 7 to 8 per cent contribution to deaths at ages 1–4 in selected runs of years during these decades, which, for instance, represents little more than half the impact of five other causes at ages 1–4 listed in the Glasgow source cited.[22] The Manchester data also appear in Davenport's figure 9, which shows that the 7 per cent figure in table 4 is mainly due to a single, moderate epidemic year in the late 1830s and that the next epidemic year occurred

[19] In Paris the trend of life expectancy was slightly rising in 1815–70, with short downward fluctuations in 1830–4 and 1845–54, coinciding with temporary fluctuations in ECMR. This does not resemble the English pattern either of deterioration in 1830–50 or of a scarlet fever plateau of high rates, supposedly driving high ECMR in the years 1850–80. Marseilles' high ECMR does not end in 1870, nor the early 1880s, but continues until 1890, unlike in Britain (Davenport's fig. 7). Its late, rapid fall coincided precisely with the city's final commitment, 'stung by a severe cholera epidemic in 1884–5', to a comprehensive sewerage system, completed in 1891–6; Preston and van de Walle, 'Urban French mortality', p. 281.

[20] Pelletier, Légaré, and Bourbeau, 'Mortality in Quebec'.

[21] van Leeuwen and Oeppen, 'Reconstructing the demographic regime', tab. 9, p. 87.

[22] Other much higher impact causes of death at ages 1–4 in Glasgow in 1836–42 were: bowel, 'decline', whooping cough, measles and smallpox; Watt, *Glasgow bills*, pp. 48–59.

in the late 1840s. This is consistent with Creighton's view and with the national data indicating that scarlet fever's new lethality did not become evident until the 1840s and that 1850–80 was the main period of impact. The Northampton data in figure 9 also confirm negligible impact before the 1840s, as does relevant research on Wigan.[23] The only evidence for a significant impact of scarlet fever as early as the 1830s comes from the US cities in figure 9. This relates principally to the city of Boston, since the graphed mortality for Philadelphia is rather moderate, while the source data for Baltimore were not considered especially robust by contemporaries.[24] But also in the 1846 primary source for the Boston data, it is cautioned of 'Scarlatina or Scarlet fever' that 'Under this name are included cases of "spotted fever", "putrid sore throat", "ulcerated sore throat", "throat distemper", "canker rash", "cynanche maligna", &c., which are supposed to be nearly synonymous'.[25]

There are, then, many evidential reasons to be cautious about placing so much confidence in scarlet fever and spontaneous changes in its virulence as to claim that it was the most significant cause of mortality trends in this period in Britain and elsewhere.

First, it is a disease whose symptoms were to varying extents shared with measles, whooping cough, and diphtheria (often termed 'croup' by the Victorians) and so statistics on its incidence, particularly before 1860, do have to be treated with considerable caution.[26] Davenport claims that scholars have tracked sequential spreads of scarlet fever from Copenhagen and port cities in Sweden to other towns and thence to rural areas from the 1840s through to the 1860s and that this is evidence of a virulent strain spreading. That may be true but also it may represent the spread of changed diagnostic or classificatory choices, given the well-documented changes in such practices in Britain during these decades (which Davenport acknowledges in the notes appended to figure 8 and to table 4).

Second, since scarlet fever was spread most intensely by aerosol exposure its transmission was magnified by the density of urban contacts and domestic over-crowding found in early Victorian towns and cities, as indicated by Woods's comparative geographic analysis. Even with no change in virulence at all, one would therefore expect to see all of these airborne diseases—and most particularly measles and scarlet fever—increasing in incidence under such changing living conditions as occurred in Britain's mushrooming towns and cities. For the same reasons, its lethality would also be expected subsequently to decline, once these urban over-crowding and sanitary problems eventually began to be alleviated at some point during the last third of the nineteenth century.

It is widely agreed that there was a reduction in scarlet fever's case-fatality rate accompanying its attenuation after 1880. Davenport claims this was due to a spontaneous change in the micro-organism's virulence. While this may be a correct surmise, it is a rather summary simplification of what is likely to have been a

[23] Crompton, 'Industrialisation', p. 212: 'scarlet fever was only recorded as a cause of around a dozen deaths in Wigan's parish registers between 1800 and 1836 and then accounted for a mere handful of deaths 1837–1840'.
[24] For instance, Joynes, 'Statistics', pp. 305–6, on 'the large number of cases in which the causes of death are "unknown"'.
[25] Shattuck, *Report*, p. 141.
[26] For informative discussions of the medical biology of scarlet fever and its historical conflation with diphtheria and other diseases, see Hardy, *Epidemic streets*, chs. 3 and 4; Mercer, *Infections*, ch. 8.

complex relationship between the 20 main types (and 60 sub-types) of this Group A streptococcus and their human hosts, themselves undergoing dramatic changes in relevant aspects of their environments, diets, and behaviour. The reasons why more virulent strains may have gained in prevalence or lethality at one time and milder ones at another time are likely to be multiple, not singular, and related to the changing environment of the hosts. There is plenty of well-documented evidence of an accumulation of factors, which combined to make urban working-class homes more sanitary, more hygienic, and less crowded during the last two decades of the century. Consequently, during the 1870s there began a reduction in the intensity of various other communicable disease insults to infants and young children which would have reduced co-morbidities—an important issue with scarlet fever—and therefore reduced case-fatality rates. Mercer has suggested that it was a prior decline in typhoid and associated enteric fevers in children aged 1–4 that enabled a subsequent decline in scarlet fever case fatalities.[27] Additionally, rising real wages facilitated the purchase of more domestic space per family; there was greater attention by local authorities to restricting over-crowding; and there were also falling urban birth-rates from the late 1870s onwards. All of these would have reduced not only the ease of transmission of scarlet fever but also the pathogenic load transmitted at each point of infection, which is agreed to be an important factor, independent of virulence issues.[28] During the 1890s there was additionally increased attention to the notification of infectious diseases and isolation of sufferers in fever hospitals, instead of leaving them to infect other family members and the wider community which, again, would have reduced co-morbidities.[29]

To conclude, the evidence put forward supporting the hypothesis that a virulent new strain of scarlet fever drove rising mortality trends across European and North American cities, as well as rural English parishes, specifically in the two decades 1830–50 is extremely weak or, on closer inspection, almost non-existent. Where age-specific mortality measures for non-British industrializing populations can be found, most of such evidence was in fact already cited by Szreter in 1997, who argued that it showed the disruptive health consequences occurring in all other well-documented cases of populations undergoing rapid economic growth.[30] However, these disruptions elsewhere did not typically happen *c*. 1825–50 as in Britain, but in later decades. Szreter also subsequently pointed out that Sweden was the only exception he could find, but that this was an exception that proved the rule, because the Swedish government was the only one which passed effective protective and preventive public health legislation in 1874 in anticipation of its industrialization, rather than in belated response to it.[31]

There is really rather little difference between Davenport and Szreter and Mooney in the positive and reliable demographic evidence presented for large

[27] Mercer, *Infections*, pp. 105–6, fig. 8.2 and tab. 8.1. The mechanism Mercer proposes is that inability to absorb key micronutrients due to enteric infections can leave young children more vulnerable to subsequent infections by other micro-organisms; and so reduction in enteric fevers and micronutrient deficiencies would have reduced the case fatality rates of scarlet fever.
[28] Breese and Breese Hall, *Disease*, pp. 41–3.
[29] Mooney, *Intrusive interventions*, ch. 3; and see Mercer, *Infections*, p. 106.
[30] Szreter, 'Economic growth', p. 694.
[31] Szreter, 'Population health approach', p. 426.

English cities. The death rates prevailing in the 1830s and 1840s are agreed to have been very high and there is just a quibble over the extent of the minor improvement which occurred in the 1850s. There is agreement that the most significant improvement did not begin to occur until the 1870s.

The rise and fall of scarlet fever mortality in Britain, *c*. 1840–90, may be due in part to changes in the incidence of the variants of the micro-organisms involved. There is some possible evidence from Boston suggesting that a virulent strain may have impacted some populations in the US already in the 1830s. However, as far as Britain is concerned, the timing of scarlet fever's impact is almost inversely correlated with the main chronological patterns of urban mortality, including the new ECMR estimates presented by Davenport. Industrial towns' mortality rose in the period 1825–50, when scarlet fever's incidence was modest, and fell back somewhat in the 1850s, when scarlet fever's impact rose to a high level. Urban mortality remained at that moderately high level in the 1860s and then fell decisively from the 1870s onwards, while scarlet fever remained at a high level of impact throughout these decades and did not fall significantly until the 1880s.

Scarlet fever was one of many early childhood communicable diseases, all of whose incidences were exacerbated by the overcrowded, insanitary, and unhygienic conditions of living faced by the expanding urban working classes in the rapidly growing towns and cities to which they increasingly migrated. As argued by Szreter in 1997, it was not until working-class men became the majority of voters, which became possible from 1869 with the passing of the Municipal Franchise Reform and Assessed Rates Acts, that municipal governments belatedly began to invest in and regulate the urban environment much more intensively than before.[32] This interpretation has since been confirmed econometrically by Aidt et al., who found that these two Acts increased the male electorate on average from 35 per cent to 57 per cent and that in the period 1868–86 it was a rise in the electorate above 40 per cent that was associated with increased expenditure on urban amenities.[33] Although these sanitary and preventive measures may have had only moderate and mediated effects on the behaviour of scarlet fever, overall it was public health measures—both their initial absence or deficiency and subsequently their increasing implementation and greater effectiveness from *c*. 1870 onwards—that had the major impacts on most communicable, sanitation, and hygiene diseases during the course of the nineteenth century.

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